Massive Troponin Release and Normal Coronary Arteries: Where Does the Truth Lie?

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CASE DESCRIPTION

A 55-year-old woman was referred to the emergency department of Padova University Hospital with acute chest pain extending up to the left shoulder. She had had fever a few days prior but no other symptoms. The patient, who was obese, smoked, and had a history of arterial hypertension, first received first-line amlodipine treatment that was interrupted because of poor compliance. No other traditional cardiovascular or thrombophilic risk factors were reported.

One hour after the onset of chest pain, the patient called the emergency service; the electrocardiogram (ECG)[3] recorded at home was unremarkable for acute ischemia (Fig. 1A). At the emergency department, after 80 min, the patient still complained of chest pain, the physical examination was negative, blood pressure was 220/110 mmHg, and heart rate was 78 beats/min. The results of the second ECG were the same as those of the first (Fig. 1B).

The initial cardiac troponin I (cTnI) concentration, obtained using a high-sensitivity assay *(1)*, was markedly increased (57 ng/L) relative to the sex-dependent 99th percentile (16 ng/L). The chest pain resolved with blood pressure normalization following intravenous nitrate administration. The patient had complained of chest pain for 100–120 min. An ECG performed after laboratory testing revealed normal left ventricular volumes and systolic function, with no cine wall motion abnormalities or other pathological signs. Contrast computerized tomography was conducted to rule out aortic dissection given the discrepancy between the type of symptoms and positive cTnI but negative ECG and echocardiogram. The examination was negative for acute aortic syndromes and no calcium was identified at the level of the coronary arteries. The patient was then transferred to the intensive coronary care unit for monitoring. The cTnI concentration on a second sample drawn 6 h later was 4017 ng/L. Chest pain had decreased, with the patient describing it as “chest discomfort”. Coronary angiography revealed normal main coronary arteries (Fig. 1C–E), without cine wall motion abnormalities at left ventricular angiography; during the procedure, neither coronary vasospasm nor embolization were documented. The cTnI peak value was 22132 ng/L on the second day, with kinetics typical of acute coronary syndrome. Contrast-enhanced cardiac magnetic resonance (CE-CMR) imaging, performed on day 6 to rule out myocarditis, showed focal akinesia of the interventricular septum between the basal and mid segments; on T2-weighted sequence, a hyperintense signal

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intensity due to myocardial edema was present in the same region of cine abnormalities (Fig. 1F). Finally, postcontrast T1-inversion recovery sequences evidenced transmural late gadolinium enhancement with an ischemic pattern and an associated hypointense core because of microvascular damage, thus indicating acute focal transmural myocardial infarction of the interventricular septum (Fig. 1G–H). In view of the CE-CMR findings, the coronary angiography was reevaluated and the small caliber of the first septal branch of the anterior descending coronary artery was confirmed (Fig. 1E). The patient was discharged on complete antiischemic and double antiplatelet drug therapy.

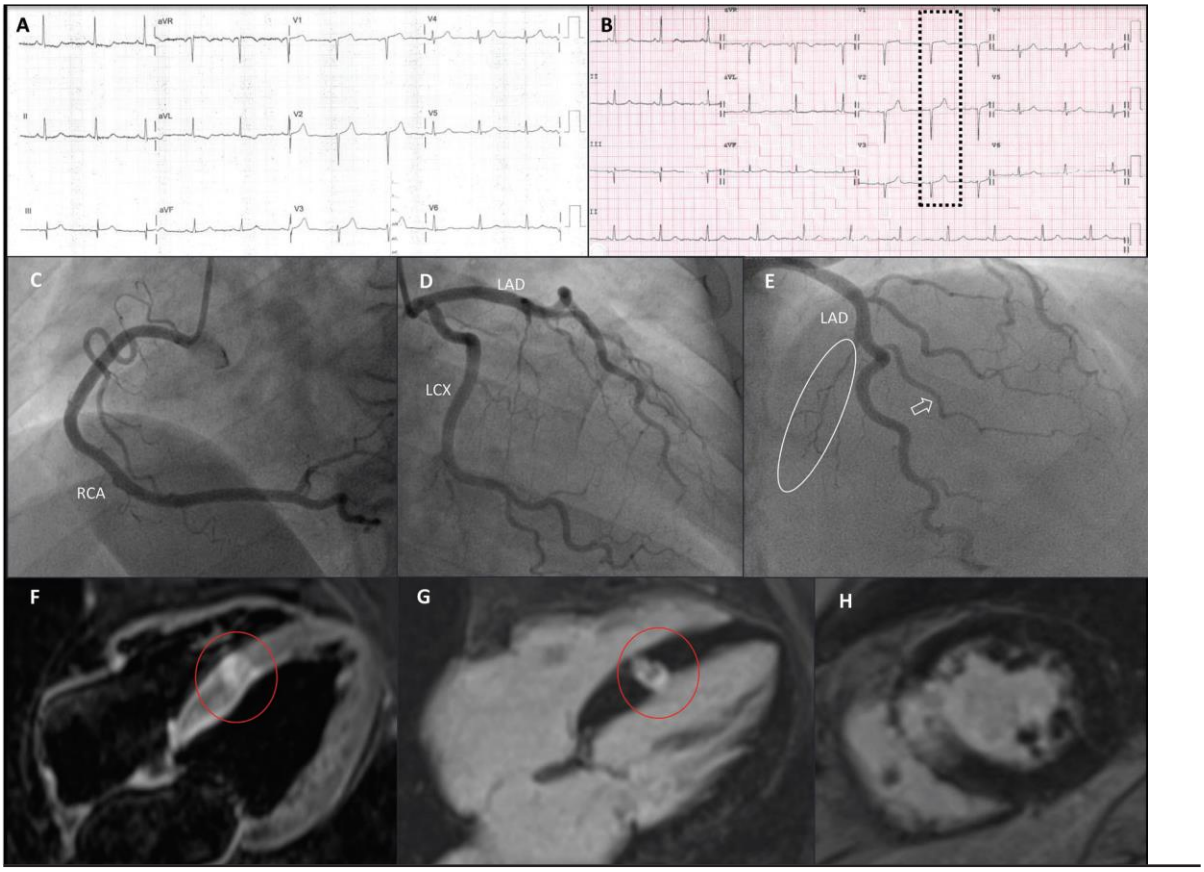

Fig. 1. Electrocardiographic and imaging findings.

First acute ECG recorded at home, unremarkable for acute ischemia (A); second ECG recorded at the emergency department (chest pain still present) not showing ST-segment elevation, but a more clear QS complex on V1–V3 (black rectangle) (B); coronary angiography showing the normal epicardial coronary artery, except the small caliber of the first septal branch of the anterior descending coronary artery (white circle on E) (C–E); T2-weighted sequence showing that the acute ischemic edema in the septum was detectable on the 4-chamber view (F); orthogonal views showing that on post-contrast CMR, the ischemic pattern of late gadolinium enhancement (white area) in the same region of the previously demonstrated edema confirmed the acute myocardial infarction (G–H); RCA, right coronary artery; LCX, left circumflex artery; LAD, left anterior descending artery.

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QUESTIONS TO CONSIDER
• What are the causes of chest pain in adults with cardiovascular risk factors?
• What are the common causes of troponin release?
• What are the kinetics typical of troponin release in acute coronary syndrome?

Reference
1. Krintus M, Kozinski M, Boudry P, Capell NE, Köller U, Lackner K, et al. European multicenter analytical evaluation of the Abbott ARCHITECT STAT high sensitive troponin I immunoassay. Clin Chem Lab Med 2014;52:1657– 65.

Final Publication and Comments

The final published version with discussion and comments from the experts will appear in the October 2017 issue of *Clinical Chemistry*. To view the case and comments online, go to http://www.clinchem.org/content/vol63/issue10 and follow the link to the Clinical Case Study and Commentaries.

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